

# Correspondence

### **Superfund and Public Health Policies: An ATSDR Response**

We read with interest the article "Public Health Policies Regarding Hazardous Waste Sites and Cigarette Smoking: An Argument by Analogy" by Legator and Strawn (p. 8). The article raises a number of important points regarding the characterization of health risks presented by hazardous waste sites. In particular, the article argues that government policies and public health practice pertaining to hazardous waste sites should somehow be developed and pursued in ways analogous to how public health agencies have reacted to the health, hazard of cigarette smoking. For reasons described in this letter, we differ with Legator and Strawn's arguments on two counts: 1) they are misinformed about how federal agencies are dealing with the health threats posed by hazardous waste sites, and 2) their proposed analogy between cigarette smoking and hazardous waste sites strikes us as being inadequate.

Turning first to the authors' attempt to develop an analogy between cigarette smoking and hazardous waste sites, it is not at all obvious to us that such a comparison can be made. We agree with Legator and Strawn that cigarette smoke contains a complex mixture of toxic substances, and acknowledge, based on our agency's experience, that some toxic waste sites release complex mixtures of substances into the environment. However, it is not true that exposure scenarios between cigarette smoking and hazardous waste sites are comparable. For example, unlike cigarette smoke, hazardous waste sites contaminate multiple environmental media in addition to air (1). Further, the degree of exposure can be vastly different, depending on such factors as proximity to hazardous waste sites, routes of exposure, and lifestyle, including smoking behav-

We also have difficulty in comparing the situation of cigarette smoking to hazardous waste sites because of the disparity in scientific knowledge. Considerable investment has been made in studying the toxicology of cigarette smoke constituents and the epidemiology of health consequences of smoking. In contrast, as pointed out by the National Research Council (2), there are an inadequate number of epidemiologic (and we would add toxicologic, as well) investigations pertaining to the health effects of hazardous wastes. Given the disparity in scientific data, how can meaningful compar-

isons between cigarette smoking and hazardous waste sites be made—especially if such comparisons, as argued by Legator and Strawn, are to be forged into public health policies?

Legator and Strawn's central thesis seems to be "The role of the public health agencies should be to identify those persons exposed to the compounds of concern. Having done so, the role of the regulatory agencies should be to eliminate the source of exposure or to relocate those persons exposed. No further assessment of the health risks is needed (our emphasis). As the principal federal public health agency tasked under the Comprehensive Environmental Response, Compensation, and Liability Act (commonly called Superfund) with determining and acting upon the effects on human health of hazardous wastes, the Agency for Toxic Substances and Disease Registry (ATSDR) is centrally involved in making the kinds of determinations advocated by Legator and Strawn. Legator and Strawn seem to suggest that current practices by EPA and ATSDR somehow differ from their central thesis. In fact, both federal agencies give special emphasis to assessing the potential of current community exposures to hazardous substances released from waste sites as well as places where emergency releases of hazardous substances have occurred. Where site conditions require, current exposures of concern are mitigated through such actions as restricting access to the site, providing alternative sources of drinking water, conducting emergency removal actions, and relocating people. Under Superfund, all of these actions have been employed by EPA, where necessary, to interdict current exposures of communities to hazardous substances released from waste sites.

We are in full agreement with the philosophical linchpin of public health practice regarding environmental hazards: prevent exposure to the hazard and thereby prevent any adverse health effects. Having said this, one is confronted with very difficult questions in terms of implementing this philosophy. How do you measure or assess exposure (especially in light of limited scientific knowledge regarding uncertainties in bioavailability and a paucity of biomarkers for most hazardous substances)? What analytic means to assess exposure are available to the public health official? When measured or assessed, how much exposure constitutes a potential health hazard? And what should be done in situations where there is information about past environmental exposures that have been interdicted? (Are there latent health effects that should be of concern to the health agency?) At the heart of these questions is how to assess or measure human exposure to toxicants in the environment.

It is ATSDR's position that exposure assessments should usually commence with an evaluation of environmental contamination levels (including an assessment of the adequacy of such data), coupled with an assessment of potential exposure pathways. From this analysis, ATSDR will conduct human exposure measurements or derive plausible estimates where that course of action is beneficial, if methods exist for measuring or estimating the levels of toxicants of concern (3). To advance the science of biological markers for use in exposure assessments, ATSDR has also supported a long-term program of work at the National Research Council. From this effort with the National Research Council have come a number of significant recommendations on biomarkers for the following toxic endpoints: reproductive, pulmonary, neurobehavioral, and immune function (4). The ATSDR is currently implementing these recommendations in its program of epidemiological investigations of communities around hazardous waste sites and other areas of pollution.

ATSDR's approach to determining who is, or has been, exposed to hazardous substances released from waste sites and other contaminated areas is contained in its public health assessment, which is an evaluation of environmental contamination data, health effects information, and community health concerns in order to determine the hazard posed by individual waste sites (5). Concerning Legator and Strawn's comment about the quality of ATSDR's health assessments, the Agency acknowledges that our public health assessments of individual waste sites were of uneven quality during a period of time when we were under severe resource constraints (6,7). However, independent peer reviews of a statistical sample of recent public health assessments, together with guidance from the ATSDR Board of Scientific Counselors, indicate ATSDR's health assessments have been improved and are of good quality. Moreover, the public health assessments of Superfund sites conducted by ATSDR and 24 state health departments have been developed into a practical instrument that points health agencies toward those public health actions (e.g., exposure assessments, epidemiologic investigations, exposure registries, surveillance) that should be pursued in communities. Even with the efforts to measure exposure, the important question about latent, adverse health effects remains unanswered. As public health professionals in environmental health committed to protect the health of communities living near hazardous waste sites, we therefore strongly disagree with the authors' statement that "No further assessment of the health risks is needed."

Legator and Strawn also make two other points to which we wish to respond. They state "If information on each site were available in sufficient detail, populations from exposed communities could be aggregated or combined. Unfortunately, the data that would help determine the multiple sites for which similar effects could be anticipated does not yet exist.' The ATSDR agrees with the approach of combining populations from sites with reasonably common characteristics; this is the exact approach taken in our National Exposure Registry program (8). As an example, the ATSDR Subregistry of Persons Exposed to Trichloroethylene consists of a registry of about 5000 persons in 13 communities. Chemical-specific exposure subregistries provide ATSDR with health information on persons with common chemical exposures and also provide a means for communicating health information back to the registrants.

In addition, more recently, ATSDR has developed the database necessary to combine site-specific information. The database is called HazDat. It contains all the environmental contamination, toxicology, and human health effects data from about 1300 Superfund sites. Recently, in conjunction with four state health departments, we conducted a study of lead exposure and toxicity in four different populations that were identified through use of HazDat. We anticipate releasing HazDat to the public later this year.

Ascertaining the dangers to public health of hazardous waste sites, together with implementing public health actions to protect against the effects of hazardous substances, is a challenging responsibility. The ATSDR's public policies and public health practices must be based on sound scientific principles and data. This must involve the communities affected by releases from waste sites and other sources of hazardous substance releases. We believe the statutory mandates in the Compre-hensive Environmental Response, Comp-ensation, and Liability Act that bear on public health are consis-

tent with sound public health practices. The translation of these mandates into actions, to some extent in ways inferred by Legator and Strawn, is ATSDR's challenge. We believe we have made progress, but much awaits.

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## Industrial Sources of Benzene Exposure?

In volume 82 of *EHP*, Wallace presented some of the results of the EPA's Total Exposure Assessment Methodology (TEAM) study in an attempt to identify the major sources of exposure to benzene (1). He contended that the results showed "... that personal activities or sources in the home far outweigh the contribution of outdoor air to human exposure to benzene" (1: 166). Two tables of statistical data were presented to demonstrate this point.

We have previously commented on the severe problems affecting the benzene data for New Jersey (2) and on the confounding effects of the apparent inversion that occurred concerning the data for Los Angeles, California (1: Tables 1 and 2; LA1). We believe that further comment is necessary regarding the North Carolina and Baltimore, Maryland, data, which are reported in the Wallace paper.

The North Carolina data presented in Wallace's Table 1 do not have an outdoor counterpart in Table 2. The reason for this is that only six fixed-site outdoor samples were obtained (3). The arithmetic mean benzene concentration of those six samples was about 19 µg/m<sup>3</sup> for both day and night, or about twice the level found in the personal air samples (4). However, neither personal exposures nor outdoor levels of benzene in North Carolina should have appeared in the paper because of the extremely high and variable levels of benzene contamination on the Tenax sampling medium. The contamination was 193 ± 216 ng benzene/tube for both personal and outdoor air samples. Regarding this contamination, the EPA report (5) says "The benzene determinations should also be viewed with suspicion. . ." We agree and believe that none of the North Carolina data should be used to draw major conclusions.

The Maryland data shown in Table 1 of Wallace's paper represents only half of the available data from that portion of the study. Wallace reports here data from the segment of the study that was downwind of an industrial district. Another segment of

Table 1. Comparison of data on levels of benzene  $(\mu g/m^3)$  from different segments of the TEAM study

	Arithmetic mean	Geometric mean	Median
Night		-	
Üpwind	9.3	2.85	5.81
Downwind	20.7	12.3	13.0
Day			
Úpwind	10.1	3.03	5.87
Downwind	16.4	8.38	11.0

the study, equal in size, from an area upwind of potential industrial sources has apparently not been reported except in the final report prepared for EPA (6). Table 1 compares data from the upwind segment of the study to data from the downwind segment of the study. Outdoor benzene levels are not reported because they were obtained by a different sampling technique.

There is no serious question about the values from the second group of data from Los Angeles (LA2) and from Antioch-Pittsburg, California (AP), but subsequent comments and conclusions regarding benzene exposure or breath differences should be reconsidered based only on results from the remaining total of 30 smokers and 89 nonsmokers. These remaining subjects can hardly be considered to be representative of the U.S. population.